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Supraventricular Arrhythmias in Children

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Sudden death may occur in children with supraventricular arrhythmias. Sick sinus syndrome, particularly if associated with tachycardia, may result in sudden death in children who have had open heart surgery and rarely in children with a normal heart. Children with supraventricular tachycardia rarely die. Only those with junctional automatic tachycardia or Wolff-Parkinson-White syndrome have died. Patients with a short anterograde refractory period may be at risk of sudden death. Surgical division of the accessory connection can prevent sudden death. Digitalis may accelerate atrioventricular (AV) conduction in patients with Wolff-Parkinson-White syndrome and, thus, should be used only after testing in the electrophysiology laboratory. Sudden death due to complete AV block should be preventable using pacemakers. Neonates with a ventricular rate less than 55 beats/min or children with a rate less than 45 beats/min should receive pacemaker therapy because of the statistical probability of death or syncope. Ventricular ectopic beats, particularly if frequent or multiform, may be an indication for pacemaker insertion. Patients with surgical complete AV block that persists for more than 7 to 10 days should receive physiologic pacemakers for the prevention of sudden death and hemodynamic benefit.

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In this review, I will discuss the role that supraventricular arrhythmias play in sudden death in children. In doing this, I am discussing the rare causes of a rare event. The arrhythmias I will include are: 1) sick sinus syndrome, both naturally occurring and acquired after open heart surgery; 2) the bradycardia-tachycardia syndrome; 3) supraventricular tachycardia, emphasizing junctional automatic tachycardia and the Wolff-Parkinson-White syndrome; and 4) complete atrioventricular (AV) block. The inclusion of complete AV block is somewhat arbitrary, but I decided to include it in this discussion so that the use of pacemakers could be kept in one place.

For each of these entities, I will discuss the etiology, pathogenesis, electrophysiologic mechanisms, natural history, association with sudden death, evaluation and treatment. The exact association of an arrhythmia with sudden death is often assumed because, by definition, sudden death is usually not monitored. The definition of sudden death is an event that occurs suddenly resulting in unconsciousness from which the patient either never recovers or is resuscitated. Thus, I will include among patients with sudden death those who lived after active resuscitation, an event that is being seen with increasing frequency.

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There is overlap between this topic and sudden death due to ventricular arrhythmias. Some cases of death in patients with primary bradyarrhythmias are due to ventricular fibrillation. In addition, death in patients with supraventricular tachycardia and Wolff-Parkinson-White syndrome is probably often due to ventricular fibrillation.

Sick Sinus Syndrome

Death from sick sinus syndrome was extremely rare in the pediatric age group until complex intraatrial surgery began to be performed (1–4). Even now, death in "pure" sick sinus syndrome is unusual, most instances occurring in patients who also had atrial tachyarrhythmias.

Isolated sick sinus syndrome. Instances of sick sinus syndrome in children who did not have congenital heart disease or cardiac surgery have been reported. At least one death in this group has been noted (1). We have reported eight instances of sick sinus syndrome in children with an otherwise normal heart and three in children without surgical treatment for congenital heart disease (5).

The etiology of these cases remains unknown. In seven of our nine symptomatic patients, abnormalities of sinus node automaticity were confirmed by an abnormal degree of overdrive suppression. In three of five, sinoatrial conduction was also prolonged. The atrial muscle refractory period was prolonged in two of eight. Atrioventricular node and His-Purkinje function was usually normal, being pro-

longed in 1 of 11 and 1 of 10, respectively. Tachyarrhythmias were inducible in 4 of 11, although none of these patients had clinical tachyarrhythmias.

Each of these patients had episodes of syncope, but none was documented to be caused by additional bradycardia. The infrequency of the episodes made their recording by ambulatory electrocardiographic monitoring unlikely. In most of these patients, ambulatory monitoring demonstrated more severe bradycardia during sleep than during the awake state. Ventricular ectopic activity was rare in this group. Exercise testing showed a blunted increase in sinus rate in most of these patients, although two had a normal response.

Role of pacemaker implantation. Each of these patients was symptomatic and 8 of 11, therefore, received a permanent pacemaker. In patients with syncope, if the heart rate decreases to less than 50 beats/min in an infant, 40 beats/min in a toddler or 30 beats/min in a teenager, it may be assumed that bradycardia is the cause of symptoms. All of our patients in this group became asymptomatic after pacemaker implantation, with the exception of one patient who also had an atrial-endocardial cushion septal defect and cardiomyopathy. After a ventricular demand pacemaker was implanted, her major symptom, syncope with grand mal seizures, was abolished. She continued to have dizzy spells. Ambulatory electrocardiographic monitoring showed that these occurred when she had sinus bradycardia and her pacemaker was turned on. Because retrograde conduction was present, "pacemaker syndrome" was suspected. An AV sequential pacemaker was implanted, and her symptoms were relieved.

Thus, cardiac pacing can relieve symptoms of syncope in patients with pure sinus node dysfunction. Whether any of these patients would have died is unclear. Very few reports of sudden death in children with a normal heart and sick sinus syndrome are available. It seems wise to use a pacemaker in these children to relieve symptoms and possibly prevent sudden death. Atrial or universal AV sequential pacemakers should be used to prevent pacemaker syndrome and provide the most physiologic response. Our policy is to use an atrial pacemaker if AV node function is normal or near normal. This is best determined by the rate of atrial pacing at which type I second degree AV block occurs. If it occurs at a rate of 110 beats/min or greater, we believe that atrial pacing is indicated. This, of course, presumes that the patient has not already had second degree or third degree AV block.

Sick sinus syndrome after cardiac surgery. Sick sinus syndrome occurs most commonly in children after cardiac surgery (6). Atrial repair of transposition of the great arteries is the most frequently associated surgical procedure, but severe cases have been found after any type of cardiac surgery using cardiopulmonary bypass. One large review of atrial septal defect repair (7) failed to find any cases of sinus node dysfunction in patients who had had their atrial septal defect repaired using hypothermia without cardiopulmonary bypass, suggesting that cannulation of the superior vena cava might be a major cause of sinus node damage. Some histologic studies have shown sutures in the sinus node and other investigations have found hemorrhage or occlusion of the sinus node artery (6,8).

Atrial septal defect repair. Bricker (personal communication), in a review of 269 cases of atrial septal defect repair, found that 7.5% of patients had symptomatic sinus node dysfunction on postoperative electrocardiography. Two had received a pacemaker, but none had died postoperatively. The prevalence of atrial tachycardias in this series was low.

Mustard operation for transposition. El-Said et al. (9), in a review of postoperative patients who had undergone the Mustard procedure, found that 2% were victims of sudden death and 3% required an implanted pacemaker. Modifications of the surgical procedure decreased the early incidence of arrhythmias, but after 5 years the incidence rate was the same. The rate of incidence remained the same in recent years when patients were operated in the first year of life.

Electrophysiologic studies show that slightly more than 50% of patients who undergo the Mustard procedure have sinus node dysfunction as indicated by decreased automaticity (Fig. 1) (10–13). A few have an abnormally prolonged sinoatrial conduction time and atrial conduction to the low lateral right atrium (13). Conduction from the sinus node to the AV node and through the AV node and His-Purkinje system to the ventricles is universally normal. Induction protocols frequently produce supraventricular tachycardias but have thus far failed to produce ventricular tachycardia, although fewer patients have been studied with ventricular protocols.

Natural history. The natural history of postoperative sinus node dysfunction seems to be progressively worse. Some sudden deaths occur in the first postoperative year, but new sudden deaths continue to occur up to 15 years postoperatively. The direct association of sudden death with sinus bradycardia remains unproven. Deaths have occurred in patients with a normally functioning pacemaker. It is likely that many deaths are due to atrial flutter with 1:1 ventricular response. A few may be due to ventricular arrhythmias.

Indications for digitalis. We have observed several instances of shock due to atrial flutter with a 1:1 response in patients who have had the Mustard procedure. One is particularly instructive (14). A 14 year old boy, 10 years after undergoing the Mustard procedure, was seen for a routine visit and found to have atrial flutter with 2:1 and 3:1 response. When informed that he was to be admitted, he began to cry and went into a 1:1 response. He was admitted to the intensive care unit within 15 minutes, but was already in pulmonary edema and required intubation. Very shortly after direct current cardioversion, the pulmonary edema froth,

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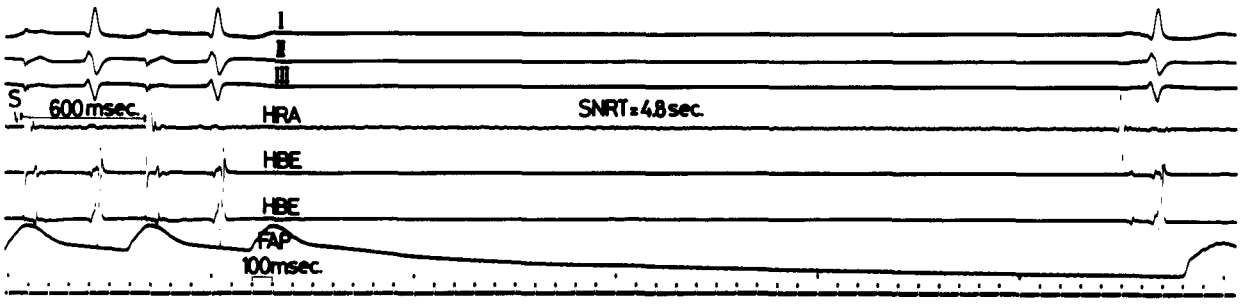

Figure 1. Electrocardiographic leads, I, II and III recorded simultaneously with high right atrial (HRA), His bundle electrograms (HBE) and femoral artery pressure (FAP). This patient had correction of a common atrium. Pacing is carried out in the high right atrium with a cycle length of 600 ms (heart rate 100 beats/min). After cessation of atrial pacing, a 4.8 second pause occurs, which is ended by a low atrial escape beat indicating severe depression of sinus node (S) and subsidiary escape pacemaker automaticity. SNRT = sinus node recovery time.

which had been abundant, disappeared and he was extubated within an hour. Because of this and other similar cases, we strongly recommend digitalization of each patient after the Mustard operation. Digitalis should be continued either indefinitely or until a 1 year or more postoperative electrophysiologic study is normal. We currently prefer continuation of digitalis indefinitely because we are not sure that our electrophysiologic techniques are infallible and because digitalis may help maintain right ventricular function.

We have not found digitalis to have a consistently negative effect on sinus node function, although in certain patients sinoatrial conduction was significantly lengthened. The clinical effect seems to be negligible. Beta-adrenergic blocking agents and type I antiarrhythmic drugs, on the other hand, can have disastrous effects. This has been documented both electrophysiologically and clinically. Our policy is to never use quinidine or beta-adrenergic blocking agents in patients who have undergone Mustard repair but who are without a pacemaker.

Role of cardiac pacemaker. The results of pacemakers in patients who have undergone the Mustard procedure have been good (15). Only 2 of 25 patients have died, both of whom had associated ventricular septal defect repair and severe bilateral cardiomyopathy. Both deaths were sudden and may have been due to tachyarrhythmias. Four patients required reoperation in the mean follow-up period of 3 years, which extended to 9 years. In two recent patients, atrial flutter was controlled using an automatic overdrive pacemaker (Cybertach).

Using our aggressive policy of digitalization, pacemaker implants and antiarrhythmic treatment, we have had only two sudden deaths in 4 years. One was in the previously described patient, and the other occurred in a patient 2 months after a Mustard operation who apparently had good sinus node function and was digitalized. He had manifested pulmonary venous obstruction in the hospital, but it had not seemed severe.

Supraventricular Tachycardia

Supraventricular tachycardia rarely causes sudden death. We have only encountered sudden death with two mechanisms of supraventricular tachycardia: junctional automatic focus tachycardia and Wolff-Parkinson-White syndrome (Table 1) (16–18). Other mechanisms of supraventricular tachycardia have not caused sudden death. There may be rare instances in which other mechanisms may lead to a relatively sudden death due to hemodynamic deterioration in patients with cardiomyopathy or congenital heart disease.

Junctional automatic tachycardia. This tachycardia is characterized by a narrow QRS complex and AV dissociation with the ventricular rate faster than the normal atrial rate (16). This tachycardia runs in families; it appears in infants and has been diagnosed in utero once. The tachycardia rate is frequently more than 200 beats/min (Fig. 2). Ventricular dysfunction and congestive heart failure occur early. This tachycardia has not been converted to sinus rhythm by any medical treatment (16,17). Patients with the mildest form may have spontaneous reversion to sinus rhythm in weeks to months. These patients invariably have a tachycardia rate of approximately 150 beats/min. Fifty percent of our medically treated patients have died suddenly. Two who were monitored at death had bradycardia immediately before death. Both were taking a beta-adrenergic blocking agent and digoxin. We have also had patients who died

Table 1. Mechanisms of Tachyarrhythmias in Children

	No. of Patients
Atrial muscle reentry	2 (4%)
AV node reentry	11 (22%)
Wolff-Parkinson-White syndrome with Kent bundle reentry	17 (34%)
Concealed Wolff-Parkinson-White syndrome	5 (10%)
Automatic ectopic atrial	5 (10%)
Ventricular reentry	10 (20%)
Total	50 (100%)

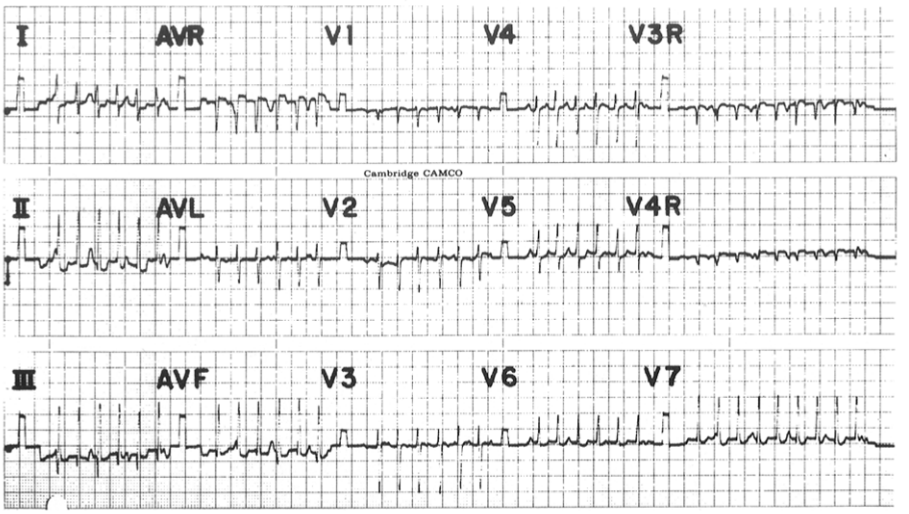

Figure 2. Fifteen lead surface electrocardiograms of junctional automatic focus tachycardia. Each lead is preceded by a calibration mark, indicating full calibration in the first six and last three leads and half calibration in the six standard precordial leads. The ventricular rate is 240 beats/min. The atrial rate is 140 beats/min and there is complete dissociation between the ventricle and the atrium. The QRS complexes are narrow and normal.

while receiving no treatment, and others who died while taking a beta-adrenergic blocking agent and amiodarone.

Pacemaker implantation and tachycardia focus ablation. Because two monitored patients died with bradycardia, we believe that the final arrhythmia may sometimes be due to complete AV block caused by a destructive lesion in the bundle of His which originally had been irritative. One pathologic study (19) also suggested His bundle abnormalities. Thus, we recommend pacemaker implantation in each patient with junctional automatic tachycardia. Because the junctional tachycardia itself has such deleterious hemodynamic effects, we also recommend destruction of the focus either surgically or by catheter ablation (Fig. 3). Catheter ablation has been successful in two of three patients, and surgical His bundle ablation has been successful in one of two. Both failures were in the patient who died during surgery at 9 days of age. Therefore, we now would implant the pacemaker at diagnosis and perform the ablation at 3 to 6 months of age after controlling congestive heart failure as best as possible.

Tachycardia Associated With Wolff-Parkinson-White Syndrome

Most tachycardias associated with Wolff-Parkinson-White syndrome involve anterograde conduction through the normal conduction system and retrograde conduction through the accessory connection (Fig. 4) (20). These probably do not cause sudden death unless they do so by precipitating atrial fibrillation. Patients with both an accessory AV connection and an atrio-His bypass tract may have such rapid supraventricular tachycardia that they have syncope but not sudden death (21). Even antidromic tachycardia, a circus movement tachycardia with anterograde conduction over the accessory connection and retrograde conduction over the bundle of His, probably does not cause sudden death.

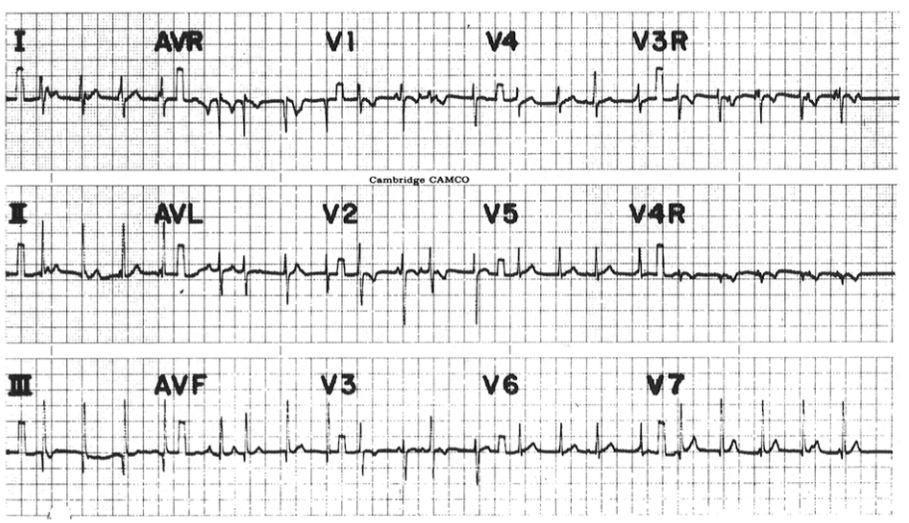

Figure 3. Postoperative 15 lead electrocardiogram of the same patient as in Figure 2 after surgical interruption of the bundle of His by cryothermia. The ventricular rate is now 120 beats/min and the atrial rate 100 beats/min. There has been no significant change in the QRS configuration, indicating that the distal bundle of His remains intact.

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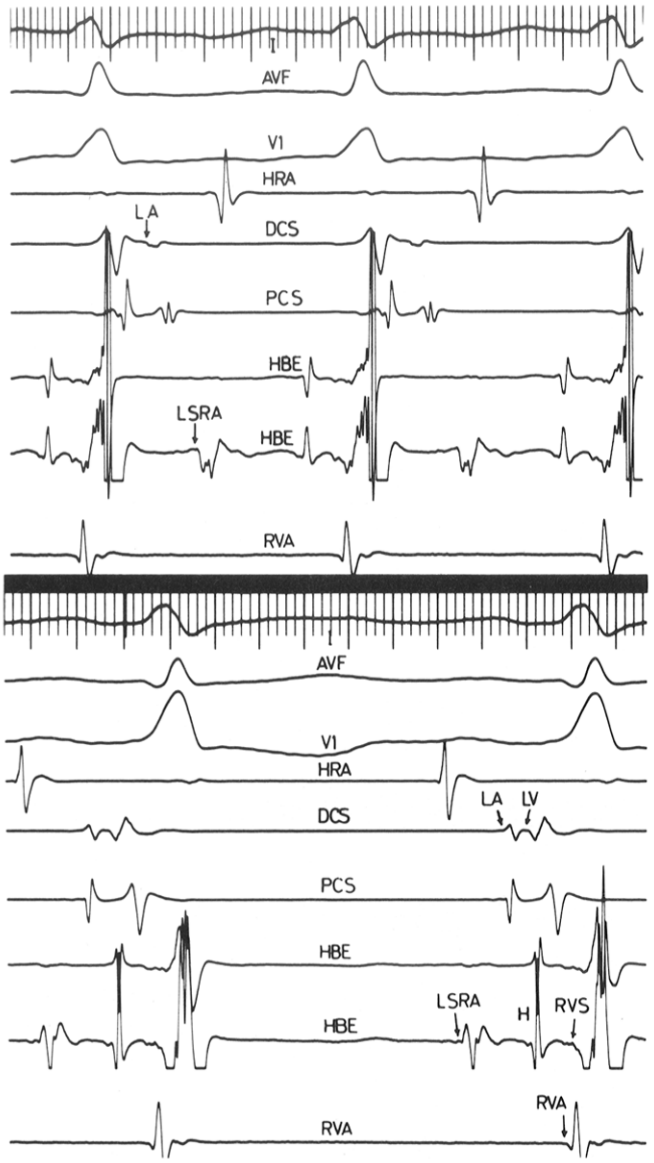


Figure 4. Top, Intracardiac recordings together with simultaneous leads I, aVF and V_1 during an episode of supraventricular tachycardia in a patient with a left-sided accessory connection. The distal coronary sinus (DCS) electrodes recording left atrial activity are activated earliest in the supraventricular tachycardia. **Bottom,** In sinus rhythm, the distal coronary sinus electrograms show early activation of the left ventricle (LV) simultaneous with depolarization of the bundle of His, indicating anterograde ventricular pre-excitation. H = His bundle; LA = left atrium; LSRA = low septal right atrium; LV = left ventricle; PCS = proximal coronary sinus; RVA = right ventricular apex; RVS = right ventricular septum; other abbreviations as in Figure 1.

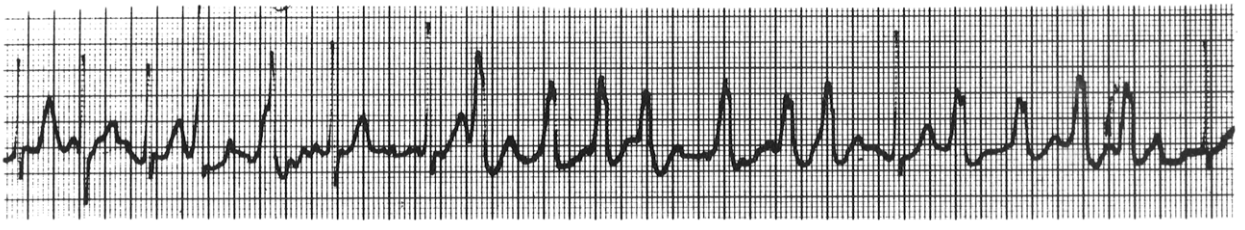
Atrial flutter or fibrillation. Atrial flutter or fibrillation in the patient with rapid conduction and a short anterograde refractory period of the accessory connection is likely to precipitate ventricular fibrillation either electrically or by hemodynamic collapse due to rapid ventricular response (Fig. 5) (22,23). Many patients who develop syncope or sudden death have never had a preceding arrhythmia. This is probably because a Kent bundle with a short anterograde refractory period presents little or no window for induction of reciprocating supraventricular tachycardia. Atrial fibrillation is uncommon in the pediatric age range.

Of our five instances of sudden death in patients with the Wolff-Parkinson-White syndrome, three were in this category. Two patients were teenagers and one was an infant. Each was resuscitated and at subsequent electrophysiologic study was found to have an anterograde refractory period between 160 and 190 ms. The infant had two Kent bundles. All of these patients underwent surgical division of their Kent bundles and they are alive and well. Other investigators (24–29) have reported sudden death in patients with accessory connections.

Role of surgical ablation of accessory pathway. What should be done with the patient found to have the Wolff-Parkinson-White syndrome on a routine electrocardiogram? We believe that such a patient should have an electrophysiologic study either transvenously or perhaps by the transesophageal route. If the anterograde refractory period is short (≤220 ms) at rest and a rapid (≤200 ms) response is found during atrial fibrillation, surgical division of the accessory connection should be considered (28,29). Only in this way can episodes of sudden death be prevented.

Role of digitalis therapy. The other two sudden deaths in our series were in patients taking digitalis for treatment of narrow QRS supraventricular tachycardia (18). One patient, an infant, had a normal heart, while the other, a teenage boy, had severe congenital heart disease. In a combined series of patients diagnosed as having the Wolff-

Figure 5. Surface electrocardiogram of atrial fibrillation in a patient with Wolff-Parkinson-White syndrome. Some beats are fully pre-excited, some beats have completely normal QRS complexes and some beats are fusion beats between pre-excitation and a normal QRS. The refractory period of the accessory connection in this patient was 280 ms.



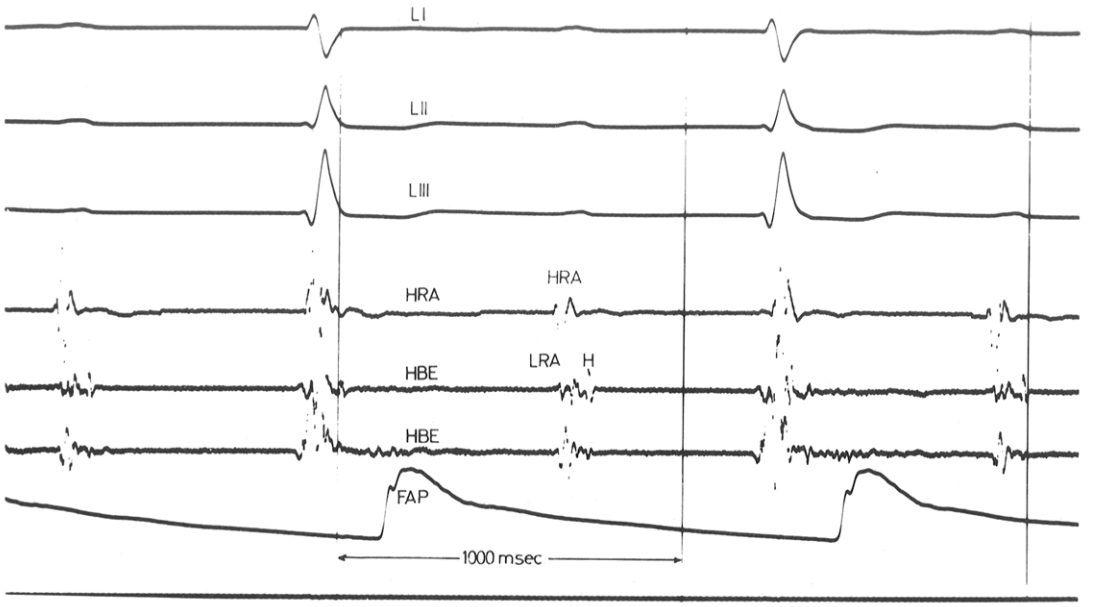


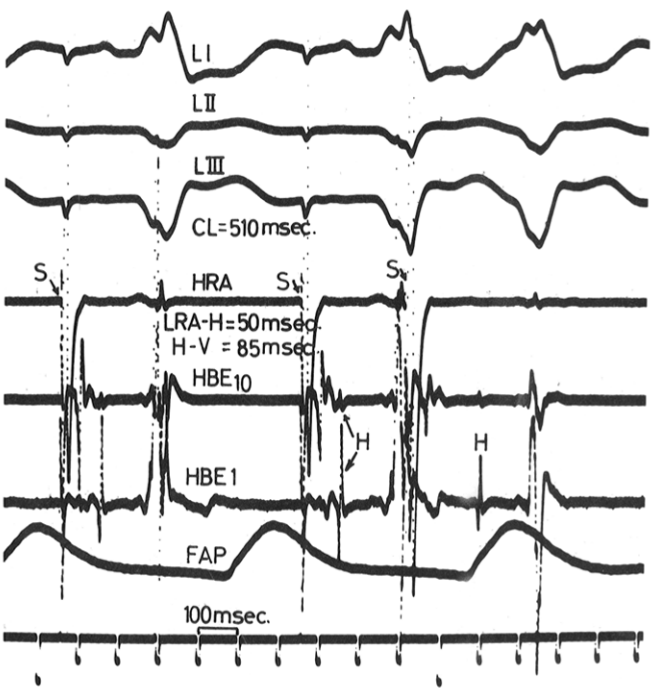
Figure 6. AV block simultaneously recorded on surface leads I, II and III, high right atrial (HRA) and two His bundle electrograms. Complete AV block is present. A His (H) spike is present after each low right atrial (LRA) depolarization. No His spike is present before the ventricular depolarization. Abbreviations as in Figure 1.

Parkinson-White syndrome at Texas Children's Hospital and Boston Children's Hospital (18), five sudden deaths were found, including those in the two patients. Each patient had been taking digoxin. One additional infant with a normal heart was included.

On the basis of these findings and those in adults suggesting that digitalis may shorten the anterograde refractory period of the accessory connection and precipitate ventricular fibrillation (30), we studied 21 patients with the Wolff-Parkinson-White syndrome before and after intravenous administration of ouabain (31). We found a shortening of the accessory connection in 43% of patients. Thus, we recommend that each patient with the Wolff-Parkinson-White syndrome and supraventricular tachycardia who needs treatment undergo an electrophysiologic study to document the functional response of the accessory connection to digitalis. If digitalis shortens the refractory period to 220 ms or less, it should not be used clinically.

Digitalis should never be given to a patient with wide QRS irregular tachycardia because the arrhythmia is probably atrial fibrillation with anterograde conduction over the Kent bundle. Verapamil has also been shown to increase the ventricular response to atrial fibrillation in children with the Wolff-Parkinson-White syndrome (32). Sudden death due to the Wolff-Parkinson-White syndrome is a largely preventable problem and should be treated as such.

Figure 7. A patient with left bundle branch block and syncopal episodes during high right atrial (HRA) pacing and induction of premature atrial stimulation. During sinus rhythm, the low right atrium to His interval (LRA-H) is 50 ms. The His to ventricle interval (H-V) is 85 ms, indicating significant prolongation of His-ventricular conduction. Because the low septal right atrium His bundle electrogram was recorded at a lower than normal value, the premature stimulus was entered after the second drive stimulus, with prolongation of the AH interval indicating normal AV node conduction. This patient's syncopal episodes were eliminated with a pacemaker. CL = cycle length; other abbreviations as in Figure 1.



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Complete AV Block

Complete AV block may be congenital, acquired after surgery or acquired without surgery. Congenital and postoperative causes are by far the most common. The likelihood of complete AV block causing sudden death is related to: 1) the ventricular escape rate, 2) the site of block, 3) its origin, and 4) associated heart disease. With current pacemaker technology, sudden death from this condition should be completely preventable.

Congenital AV block. Congenital complete AV block in infants is unlikely to produce sudden death. There are, however, several subsets of patients at risk for sudden death (33–36). Neonates with ventricular escape rates less than 55 beats/min are at higher risk of sudden death, even in the first 24 hours of life. This event cannot be predicted by the overall condition of the infant. Even those who appear well may die suddenly after a decrease in their pacemaker rate. Temporary pacing is indicated immediately in this situation, and a permanent pacemaker should be implanted the next day. Physiologic dual chamber pacemakers are now small enough to use in the normal-sized newborn. To facilitate treatment, each fetus detected as having complete AV block antenatally should be transferred and delivered in a center with a pediatric cardiologist and pacemaker implant facilities.

Infants with significant associated congenital heart defects tolerate AV block less well and die suddenly more frequently (33,34). They should receive pacemaker therapy if their ventricular escape rate is less than 65 beats/min or if they have moderate or severe congestive heart failure.

Death from congenital complete AV block after the first year of life is rare but does occur, usually in relation to a major stress. We have found that a ventricular rate less than 45 beats/min is associated with developing syncope and may thus be related to sudden death (37). We therefore consider an escape rate of 45 beats/min or less to be an indication for a pacemaker. Complex ventricular ectopic activity is another suspected cause of death in congenital complete AV block (38). We have found that implantation of an atrial synchronous pacemaker will abolish premature ventricular complexes in most such patients.

A wide QRS escape rhythm is thought to represent a poor prognosis in any patient with congenital complete AV block (39). This is usually associated with block below the His potential if intracardiac recordings are performed (Fig. 6). Block within the His/split His potential may be related to a poor prognosis, but this is not yet proven. These patients are probably identified by their slow escape rate. A prolonged corrected QT interval has also been found to be associated with an increased incidence of sudden death (39).

In a European study (40), 2 of 44 patients with congenital complete AV block died during a 12 year follow-up study and 9 required a pacemaker. In their study of sudden death in children, Lambert et al. (41) found that 4% of sudden deaths in patients without heart surgery occurred in those with congenital complete AV block. Although congenital complete AV block is an infrequent cause of death, it should be one of the more easily preventable.

Postoperative AV block. Patients with postoperative complete AV block are at high risk of sudden death (42). We detected a subgroup with block above the His bundle and a ventricular escape rate of greater than 60 beats/min who did not have sudden death (43). Unfortunately, these patients tended to develop congestive heart failure over a period of years. We, therefore, recommend permanent dual chamber atrial tracking pacemakers for each patient whose block persists for 7 days or more.

Acquired AV block. Children with acquired complete AV block from other causes are also at high risk of death (Fig. 7). If the block persists, they probably should be treated as are those with surgical block, although data on this subject are scanty.

Conclusions

We currently possess most of the knowledge and techniques necessary to prevent sudden death from supraventricular arrhythmias in children. This is in contradistinction to many of the other topics discussed in this Conference, such as sudden infant death syndrome. In the area of supraventricular arrhythmias the need is for education and demonstration of the results of treatment of these arrhythmias. Improvements can also be made if we can treat abnormalities such as the Wolff-Parkinson-White syndrome by catheter techniques rather than open heart surgery. Further improvements in the size of pacemakers and their leads should also be possible.

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